Connecting via Winsock to STN

Welcome to STN International! Enter x:x

LOGINID:ssspta1649jxm

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

* * * * * * * * * * Welcome to STN International * * * * * * * * * *

NEWS	1		Web Page URLs for STN Seminar Schedule - N. America
NEWS	2		"Ask CAS" for self-help around the clock
NEWS	3	SEP 09	CA/CAplus records now contain indexing from 1907 to the present
NEWS	4	Jul 15	Data from 1960-1976 added to RDISCLOSURE
NEWS	5	Jul 21	Identification of STN records implemented
NEWS	6	Jul 21	Polymer class term count added to REGISTRY
NEWS	7	Jul 22	INPADOC: Basic index (/BI) enhanced; Simultaneous Left and Right Truncation available
NEWS	8	AUG 05	New pricing for EUROPATFULL and PCTFULL effective August 1, 2003
NEWS	9	AUG 13	Field Availability (/FA) field enhanced in BEILSTEIN
NEWS	10	AUG 15	PATDPAFULL: one FREE connect hour, per account, in September 2003
NEWS	11	AUG 15	PCTGEN: one FREE connect hour, per account, in September 2003
NEWS	12	AUG 15	RDISCLOSURE: one FREE connect hour, per account, in September 2003
NEWS	13	AUG 15	TEMA: one FREE connect hour, per account, in September 2003
NEWS	14	AUG 18	Data available for download as a PDF in RDISCLOSURE
NEWS	15	AUG 18	Simultaneous left and right truncation added to PASCAL
NEWS	16	AUG 18	FROSTI and KOSMET enhanced with Simultaneous Left and Righ Truncation
NEWS	17	AUG 18	Simultaneous left and right truncation added to ANABSTR
NEWS	18	SEP 22	DIPPR file reloaded
NEWS	19	SEP 25	INPADOC: Legal Status data to be reloaded
NEWS	20	SEP 29	DISSABS now available on STN

NEWS EXPRESS	April 4 CURRENT WINDOWS VERSION IS V6.01a, CURRENT MACINTOSH VERSION IS V6.0b(ENG) AND V6.0Jb(JP), AND CURRENT DISCOVER FILE IS DATED 01 APRIL 2003
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS INTER	General Internet Information
NEWS LOGIN	Welcome Banner and News Items
NEWS PHONE	Direct Dial and Telecommunication Network Access to STN
NEWS WWW	CAS World Wide Web Site (general information)

Enter NEWS followed by the item number or name to see news on that specific topic.

All use of STN is subject to the provisions of the STN Customer agreement. Please note that this agreement limits use to scientific research. Use for software development or design or implementation of commercial gateways or other similar uses is prohibited and may result in loss of user privileges and other penalties.

* * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 13:29:33 ON 29 SEP 2003

=> file medline biosis embase caplus
COST IN U.S. DOLLARS

	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 13:29:43 ON 29 SEP 2003

FILE 'BIOSIS' ENTERED AT 13:29:43 ON 29 SEP 2003
COPYRIGHT (C) 2003 BIOLOGICAL ABSTRACTS INC.(R)

FILE 'EMBASE' ENTERED AT 13:29:43 ON 29 SEP 2003
COPYRIGHT (C) 2003 Elsevier Inc. All rights reserved.

FILE 'CAPLUS' ENTERED AT 13:29:43 ON 29 SEP 2003
USE IS SUBJECT TO THE TERMS OF YOUR STN CUSTOMER AGREEMENT.
PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
COPYRIGHT (C) 2003 AMERICAN CHEMICAL SOCIETY (ACS)

=> s (voltage (s) sensitive (s) dye) (p) (express? (s) ion (s) channel)
L1 31 (VOLTAGE (S) SENSITIVE (S) DYE) (P) (EXPRESS? (S) ION (S) CHANNEL)

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2 18 DUP REM L1 (13 DUPLICATES REMOVED)

=> d l2 total ibib kwic

L2 ANSWER 1 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 1
ACCESSION NUMBER: 2003:412775 BIOSIS
DOCUMENT NUMBER: PREV200300412775
TITLE: Nonlinear effects in subthreshold virtual electrode polarization.
AUTHOR(S): Sambelashvili, Aleksandre T.; Nikolski, Vladimir P.; Efimov, Igor R. (1)
CORPORATE SOURCE: (1) Case Western Reserve Univ., 10900 Euclid Ave., Wickenden Bldg., Rm. 520, Cleveland, OH, 44106-7207, USA: ire@cwru.edu USA
SOURCE: American Journal of Physiology, (June 2003, 2003) Vol. 284, No. 6 Part 2, pp. H2368-H2374. print.
ISSN: 0002-9513.
DOCUMENT TYPE: Article
LANGUAGE: English
AB. . . . We studied cardiac membrane polarization produced by subthreshold stimuli in 1) rabbit ventricular muscle using high-resolution fluorescent imaging with the **voltage-sensitive dye** pyridinium 4-(2-(6-(dibutylamino)-2-naphthalenyl)-ethenyl)-1-(3-sulfopropyl)hydroxide (di-4-ANEPPS) and 2) an active bidomain model with Luo-Rudy **ion channel** kinetics. Both in vitro and in numero models show that the common dog-bone-shaped VEP is present at any stimulus strength during both systole and diastole. Diastolic subthreshold VEPs exhibited nonlinear properties that were **expressed** in time-dependent asymmetric reversal of membrane polarization with respect to stimulus polarity. The bidomain model reveals that this asymmetry is due to nonlinear properties of the inward rectifier potassium current. Our results suggest that active **ion channel** kinetics modulate the transmembrane polarization pattern that is predicted by the linear bidomain model of cardiac syncytium.

L2 ANSWER 2 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN

ACCESSION NUMBER: 2003:213163 BIOSIS
DOCUMENT NUMBER: PREV200300213163
TITLE: Measurement of membrane potential from colonies of HEK293 cells transiently **expressing ion channels** by use of **voltage-sensitive** fluorescent **dye**.
AUTHOR(S): Hotta, Aya (1); Ohya, Susumu (1); Muraki, Katsuhiko (1); Imaizumi, Yuji (1)
CORPORATE SOURCE: (1) Dept. Mol. Cell. Pharmacol., Grad. Sch. Pharm. Sci., Nagoya City Univ., Nagoya, 467-8603, Japan Japan
SOURCE: Journal of Pharmacological Sciences, (2003) Vol. 91, No. Supplement I, pp. 245P. print.
Meeting Info.: 76th Annual Meeting of the Japanese Pharmacological Society Fukuoka, Japan March 24-26, 2003 Japanese Pharmacological Society
. ISSN: 1347-8613.
DOCUMENT TYPE: Conference
LANGUAGE: English
TI Measurement of membrane potential from colonies of HEK293 cells transiently **expressing ion channels** by use of **voltage-sensitive** fluorescent **dye**.

L2 ANSWER 3 OF 18 CAPLUS COPYRIGHT 2003 ACS on STN
ACCESSION NUMBER: 2002:522155 CAPLUS
DOCUMENT NUMBER: 137:91389
TITLE: cDNAs encoding mammalian taste receptor cell- specific ion channel subunits and screening for effectors of taste signaling
INVENTOR(S): Zuker, Charles S.; Zhang, Yifeng
PATENT ASSIGNEE(S): The Regents of the University of California, USA
SOURCE: PCT Int. Appl., 306 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2002054069	A1	20020711	WO 2001-US49808	20011226
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2002164645	A1	20021107	US 2001-26188	20011221

PRIORITY APPLN. INFO.: US 2000-259379P P 20001229
US 2001-26188 A 20011221
REFERENCE COUNT: 5 THERE ARE 5 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT
AB The invention includes nucleic acid and amino acid sequences of a mouse, human and rat taste cell-specific **ion channel** subunit that is specifically **expressed** in taste cells. Also provided are antibodies to such subunits, methods of detecting such nucleic acids and proteins, and methods of screening for modulators of taste cell specific ion channel subunit signaling. More specifically, taste cell-specific ion channels modulate the transmembrane Ca2+ ion flux which may be monitored by **voltage** clamp assays, patch clamp assays, radiolabeled ion flux assays or fluorescence assays using ion

sensitive dyes.

L2 ANSWER 4 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 2
ACCESSION NUMBER: 2002:205100 BIOSIS
DOCUMENT NUMBER: PREV200200205100
TITLE: Transgenic mice expressing a pH and Cl- sensing yellow-fluorescent protein under the control of a potassium channel promoter.
AUTHOR(S): Metzger, Friedrich; Repunte-Canonigo, Vez; Matsushita, Shinichi; Akemann, Walther; Diez-Garcia, Javier; Ho, Chi Shun; Iwasato, Takuji; Grandes, Pedro; Itohara, Shigeyoshi; Joho, Rolf H.; Knopfel, Thomas (1)
CORPORATE SOURCE: (1) Laboratory for Neuronal Circuit Dynamics, Brain Science Institute, RIKEN, 2-1 Hirosawa, Wako-shi, Saitama, 351-0198: knopfel@brain.riken.go.jp Japan
SOURCE: European Journal of Neuroscience, (January, 2002) Vol. 15, No. 1, pp. 40-50. http://www.blackwell-science.com/cgilib/jnlpage.asp?Journal=ejn&File=ejn. print.
ISSN: 0953-816X.
DOCUMENT TYPE: Article
LANGUAGE: English
AB. . . years a variety of genetically encodable optical probes that monitor physiological parameters such as local pH, Ca2+, Cl-, or transmembrane **voltage** have been developed. These sensors are based on variants of green-fluorescent protein (GFP) and can be synthesized by mammalian cells. . . after transfection with cDNA. To use these sensor proteins in intact brain tissue, specific promoters are needed that drive protein **expression** at a sufficiently high **expression** level in distinct neuronal subpopulations. Here we investigated whether the promoter sequence of a particular potassium **channel** may be useful for this purpose. We produced transgenic mouse lines carrying the gene for enhanced yellow-fluorescent protein (EYFP), a yellow-green pH- and Cl- **sensitive** variant of GFP, under control of the Kv3.1 K+ **channel** promoter (pKv3.1). Transgenic mouse lines displayed high levels of EYFP **expression**, identified by confocal microscopy, in adult cerebellar granule cells, interneurons of the cerebral cortex, and in neurons of hippocampus and thalamus. Furthermore, using living cerebellar slices we demonstrate that **expression** levels of EYFP are sufficient to report intracellular pH and Cl- concentration using imaging techniques and conditions analogous to those used with conventional **ion-sensitive dyes**. We conclude that transgenic mice **expressing** GFP-derived sensors under the control of cell-type specific promoters, provide a unique opportunity for functional characterization of defined subsets of. . .

L2 ANSWER 5 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2001:540671 BIOSIS
DOCUMENT NUMBER: PREV200100540671
TITLE: Design and characterization of a DNA encoded voltage sensitive fluorescent protein.
AUTHOR(S): Knopfel, T. (1); Repunte-Canonigo, V. (1); Raj, C. D. (1); Sakai, R. (1)
CORPORATE SOURCE: (1) Laboratory for Neuronal Circuit Dynamics, Brain Science Institute, RIKEN, Wako-shi Japan
SOURCE: Society for Neuroscience Abstracts, (2001) Vol. 27, No. 2, pp. 1583. print.
Meeting Info.: 31st Annual Meeting of the Society for Neuroscience San Diego, California, USA November 10-15, 2001
ISSN: 0190-5295.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English

AB. . . suggested as a promising approach to investigate the multineuronal representation of information processing in brain tissue. However, intrinsic or extrinsic **dye**-mediated optical signals are often of limited use due to their slow response dynamics, low effective sensitivity, toxicity or undefined cellular origin. Protein-based and DNA encoded **voltage** sensors could overcome these limitations. Here we report the design and generation of a **voltage sensitive** fluorescent protein (VSFP1) consisting of a **voltage** sensing domain of a potassium **channel** and a pair of cyan and yellow mutants of green fluorescent protein (GFP). Modulation of fluorescence intensity by membrane potential was investigated in **voltage**-clamped HEK cells **expressing** VSFP1. Depolarizing **voltage** jumps resulted in an increase in the emission by YFP (>530 nm) with excitation of CFP (432 nm) while hyperpolarization of the membrane resulted in a decrease in fluorescence output. The current-to-**voltage** relationship of HEK cells **expressing** VSFP1 did not differ from that of untransfected cells demonstrating that VSFP1 did not form functional **ion**-conducting **channels**. The relationship between **voltage** change and fluorescence change was close to linear (r=0.99) with a slope of 1.8+-0.1%/100 mV (n=11 cells). In parallel measurements using the prototypic conventional **voltage sensitive dye** di-4-ANEPPS, we obtained a sensitivity of -5.3+-0.3%/100 mV from clean HEK cell membranes. The optical signals responded in the millisecond time scale of fast electrical signaling and are large enough to allow monitoring **voltage** changes at the single cell level.

L2 ANSWER 6 OF 18 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2001164445 MEDLINE
DOCUMENT NUMBER: 21163415 PubMed ID: 11265727
TITLE: Cellular basis for dispersion of repolarization underlying reentrant arrhythmias.
AUTHOR: Akar F G; Laurita K R; Rosenbaum D S
CORPORATE SOURCE: Department of Medicine, Heart and Vascular Research Center, Case Western Reserve University, Cleveland, OH 44109-1998, USA.
SOURCE: JOURNAL OF ELECTROCARDIOLOGY, (2000) 33 Suppl 23-31. Ref: 26
Journal code: 0153605. ISSN: 0022-0736.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200104
ENTRY DATE: Entered STN: 20010502
Last Updated on STN: 20010502
Entered Medline: 20010426
AB Substantial heterogeneity in **ion channel** density and **expression** exists in cells isolated from various regions of the heart. Cell-to-cell coupling in the intact heart, however, is expected to attenuate the functional **expression** of the **ion channel** heterogeneities. Due to limitations of conventional electrophysiological recording techniques, the extent to which cellular electrical heterogeneities are functionally present in intact myocardium remains unknown. High-resolution optical mapping with **voltage-sensitive dyes** was used to measure transepicardial and transmural repolarization gradients in the Langendorff perfused guinea pig ventricle and the canine wedge. . .

L2 ANSWER 7 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 2001:88917 BIOSIS
DOCUMENT NUMBER: PREV200100088917

TITLE: Mechanisms of hypoxic excitation of vasomotor neurons of rostral ventrolateral medulla.
AUTHOR(S): Wang, G. (1); Zhou, P.; Repucci, M.; Reis, D. J.
CORPORATE SOURCE: (1) Weill Med. Coll. of Cornell Univ., New York, NY USA
SOURCE: Society for Neuroscience Abstracts, (2000) Vol. 26, No. 1-2, pp. Abstract No.-443.11. print.
Meeting Info.: 30th Annual Meeting of the Society of Neuroscience New Orleans, LA, USA November 04-09, 2000 Society for Neuroscience
. ISSN: 0190-5295.
DOCUMENT TYPE: Conference
LANGUAGE: English
SUMMARY LANGUAGE: English
AB. . . excited by hypoxia, initiating patterned autonomic responses of O2-conserving (diving) reflex. The O2-sensing in peripheral chemoreceptor cells is associated with O2-**sensitive** K+ **channel** activity. We investigated whether hypoxic excitation of RVLM neurons results from activation or inhibition of O2-**sensitive**, **voltage**-gated **ion channels**. RVLM neurons from 3-11 day old rat pups were retrogradely labeled with rhodamine-labeled **dyes** injected into the T2-T4 spinal segment. Brainstem slices (150-200mu) were obtained and RVLM neurons identified under epifluorescence. The labeled RVLM. . . 2.2 mV (n=3, p<0.05) without any SD by 125 muM NaCN. To determine if this hypoxic effect is related to O2-**sensitive** **voltage**-gated **ion channels** , the **ion** currents of RVLM neurons were recorded using the whole-cell **voltage**-clamp. While the Na+, A-type K+ and Ca++ currents were not significantly affected by NaCN, a sustained outward K+ current was. . . controls to 947 +- 109.8 pA of NaCN-treated neurons (n=4, p<0.01). Post-recording single cell RT-PCR was also conducted. RVLM neurons **expressed** TH and O2-**sensitive**, **voltage** -gated K+ **channels** Kv2.1 and Kv 3.1. These results suggest that inhibition of O2-**sensitive** K+ **channels** might contribute to hypoxic excitation of RVLM neurons.

L2 ANSWER 8 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 4
ACCESSION NUMBER: 1999:374940 BIOSIS
DOCUMENT NUMBER: PREV199900374940
TITLE: Modulation of glioma cell migration and invasion using Cl- and K+ ion channel blockers.
AUTHOR(S): Soroceanu, Liliana; Manning, Timothy J., Jr.; Sontheimer, Harald (1)
CORPORATE SOURCE: (1) 1719 6th Avenue South CIRC 545, Birmingham, AL, 35294-0021 USA
SOURCE: Journal of Neuroscience, (July 15, 1999) Vol. 19, No. 14, pp. 5942-5954.
ISSN: 0270-6474.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB. . . Mechanisms that allow glioma cells to disseminate, migrating through the narrow extracellular brain spaces are poorly understood. We recently demonstrated **expression** of large **voltage**-dependent chloride (Cl-) currents, selectively **expressed** by human glioma cells in vitro and in situ (Ullrich et al., 1998). Currents are **sensitive** to several Cl- **channel** blockers, including chlorotoxin (Ctx), (Ullrich and Sontheimer, 1996; Ullrich et al., 1996), tetraethylammonium chloride (TEA), and tamoxifen (Ransom and Sontheimer, 1998). Using Transwell migration assays, we show that blockade of glioma Cl- **channels** specifically inhibits tumor cell migration in a dose-dependent manner. Ctx (5 muM), tamoxifen (10 muM), and TEA (1 mM) also. . . brain aggregates, used as an in vitro model to assess tumor invasiveness. Anion replacement studies suggest that permeation of

chloride **ions** through glioma chloride **channel** is obligatory for cell migration. Osmotically induced cell swelling and subsequent regulatory volume decrease (RVD) in cultured glioma cells were. . . in glioma cells were inhibited by 5 muM Ctx, 10 muM tamoxifen, and 1 mM TEA, as determined using the Cl-**sensitive** fluorescent **dye** 6-methoxy-N-ethylquinolinium iodide. Collectively, these data suggest that chloride **channels** in glioma cells may enable tumor invasiveness, presumably by facilitating cell shape and cell volume changes that are more conducive. . .

L2 ANSWER 9 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 5
ACCESSION NUMBER: 1999:351188 BIOSIS
DOCUMENT NUMBER: PREV199900351188
TITLE: Block by ruthenium red of cloned neuronal voltage-gated calcium channels.
AUTHOR(S): Cibulsky, Susan M.; Sather, William A. (1)
CORPORATE SOURCE: (1) Neuroscience Center, B-138, University of Colorado Health Sciences Center, 4200 E. 9th Ave., Denver, CO, 80262 USA
SOURCE: Journal of Pharmacology and Experimental Therapeutics, (June, 1999) Vol. 289, No. 3, pp. 1447-1453. ISSN: 0022-3565.
DOCUMENT TYPE: Article
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The **dye** ruthenium red (RuR) has diverse experimental uses, including block of **ion channels**. RuR is a well described antagonist of one class of intracellular Ca2+ release **channels**, the ryanodine receptors, but recently this compound has also been identified as a putative blocker of **voltage**-gated calcium **channels** of the surface membrane involved in neurotransmitter release. Using electrophysiological methods, we have studied the action of RuR upon pure populations of neuronal **voltage**-gated **ion channels** heterologously **expressed** in Xenopus laevis oocytes. All four **channel** types studied, including class A (P/Q-type), class B (N-type), class C (L-type), and class E **channels**, are **sensitive** to RuR, with IC50 values ranging from 0.7 to 67.1 muM. Block of class C and class E **channels** most likely results from 1:1 binding of ruthenium red at a site in the extracellular entrance to the pore, resulting in obstruction of permeant **ion** flux through these **channels** . The mechanism of block of classA and class B **channels** is more complex, requiring binding of more than one molecule of RuR per **channel**.

L2 ANSWER 10 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 6
ACCESSION NUMBER: 1998:483420 BIOSIS
DOCUMENT NUMBER: PREV199800483420
TITLE: Rapid Ca2+ entry through Ca2+-permeable AMPA/kainate channels triggers marked intracellular Ca2+ rises and consequent oxygen radical production.
AUTHOR(S): Carriedo, Sean G.; Yin, Hong Zhen; Sensi, Stefano L.; Weiss, John H. (1)
CORPORATE SOURCE: (1) Dep. Neurology, Univ. California, Irvine, Irvine, CA 92697-4292 USA
SOURCE: Journal of Neuroscience, (Oct. 1, 1998) Vol. 18, No. 19, pp. 7727-7738. ISSN: 0270-6474.
DOCUMENT TYPE: Article
LANGUAGE: English
AB The widespread neuronal injury that results after brief activation of highly Ca2+-permeable NMDA **channels** may, in large part, reflect

mitochondrial Ca2+ overload and the consequent production of injurious oxygen radicals. In contrast, AMPA/kainate receptor. . . studies have not found evidence of comparable oxygen radical production. Subsets of central neurons, composed mainly of GABAergic inhibitory interneurons, **express** AMPA/kainate **channels** that are directly permeable to Ca2+ **ions**. Microfluorometric techniques were performed by using the oxidation-**sensitive dye** hydroethidine (HEt) to determine whether the relatively rapid Ca2+ flux through AMPA/kainate **channels expressed** on GABAergic neurons results in oxygen radical production comparable to that triggered by NM DA. Consistent with previous studies, NMDA. . . triggered increases in fluorescence in most cultured cortical neurons, whereas high K+ (50 mM) exposures (causing depolarization-induced Ca2+ influx through **voltage-sensitive** Ca2+ **channels**) caused little fluorescence change. In contrast, kainate exposure caused fluorescence increases in a distinct subpopulation of neurons; immunostaining for glutamate. . . oxygen radical production paralleled the effect of these exposures on intracellular Ca2+ levels when they were monitored with the low-affinity Ca2+-**sensitive dye** fura-2FF, but not with the high-affinity **dye** fura-2. Inhibition of mitochondrial electron transport with CN- or rotenone almost completely blocked kainate-triggered oxygen radical production. Furthermore, antioxidants attenuated. . . resulting from brief exposures of NMDA or kainate. Thus, as with NMDA receptor activation, rapid Ca2+ influx through Ca2+-permeable AMPA/kainate **channels** also may result in mitochondrial Ca2+ overload and consequent injurious oxygen radical production.

L2 ANSWER 11 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1998:484015 BIOSIS
DOCUMENT NUMBER: PREV199800484015
TITLE: Voltage-activated currents in identified giant interneurons isolated from adult crickets Gryllus bimaculatus.
AUTHOR(S): Kloppenburg, Peter; Hoerner, Michael (1)
CORPORATE SOURCE: (1) Inst. Zool. Anthropol., Dep. Cell Biol., Univ. Goettingen, Berliner Strasse 28, D-37073 Goettingen Germany
SOURCE: Journal of Experimental Biology, (Sept., 1998) Vol. 201, No. 17, pp. 2529-2541.
ISSN: 0022-0949.
DOCUMENT TYPE: Article
LANGUAGE: English
AB. . . bodies was established. Prior to cell dissociation, the giant interneurons were backfilled through their axons in situ with a fluorescent **dye** (dextran tetramethylrhodamine). In primary cell cultures, the cell bodies of giant interneurons were identified among a population of co-cultured neurons. . . their red fluorescence. Action potentials were recorded from the cell bodies of the cultured interneurons suggesting that several types of **voltage**-activated **ion channels** exist in these cells. Using **voltage**-clamp recording techniques, four **voltage**-activated currents were isolated and characterized. The giant interneurons **express** at least two distinct K+ currents: a transient current that is blocked by 4-aminopyridine (4 X 10-3 mol-1) and a. . . partially blocked by tetraethylammonium (3 X 10-2 mol-1) and quinidine (2 X 10-4 mol-1). In addition, a transient Na+ current **sensitive** to 10-7 mol l-1 tetrodotoxin and a Ca2+ current blocked by 5 X 10-4 mol l-1 CdCl2 have been characterized.. . .

L2 ANSWER 12 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 7
ACCESSION NUMBER: 1996:37100 BIOSIS
DOCUMENT NUMBER: PREV199698609235
TITLE: Activation of nicotinic acetylcholines receptors expressed in quail fibroblasts: Effects on intracellular calcium.

AUTHOR(S): Cross, K. M. L. (1); Jane, S. D.; Wild, A. E.; Foreman, R. C.; Chad, J. E.
CORPORATE SOURCE: (1) Dep. Physiol. Pharmacol., University Southampton, Southampton SO16 7PX UK
SOURCE: British Journal of Pharmacology, (1995) Vol. 116, No. 7, pp. 2838-2844.
ISSN: 0007-1188.
DOCUMENT TYPE: Article
LANGUAGE: English
AB 1 The aim of these experiments was to determine the ability of the muscle-type nicotinic acetylcholine receptor (AChR) stably **expressed** in quail fibroblasts (QF18 cells) to elevate intracellular calcium ((Ca-2+)-i) upon activation. Ratiometric confocal microscopy, with the calcium-**sensitive** fluorescent **dye** Indo-1 was used. 2 Application of the nicotinic agonist, suberyldicholine (SDC), to the transfected QF18 cells caused an increase in. . . were blocked by prior application of alpha-bungarotoxin (200 nM), by the addition of Ca-2+ (100 mu-M), by removal of Na+ **ions** from the extracellular solution, or by the **voltage-sensitive** calcium **channel** blockers nifedipine and omega-conotoxin, which act with IC-50 values of 100 nM and 100 pM respectively. 5 We conclude that activation of the nicotinic AChRs leads to a Na+-dependent depolarization and hence activation of endogenous **voltage-sensitive** Ca-2+ **channels** in the plasma membrane and an increase in (Ca-2+)-i. There is no significant entry of Ca-2+ through the nicotinic receptor. . .

L2 ANSWER 13 OF 18 MEDLINE on STN DUPLICATE 8
ACCESSION NUMBER: 96342976 MEDLINE
DOCUMENT NUMBER: 96342976 PubMed ID: 8741761
TITLE: Zn(2+) permeates Ca(2+) permeable AMPA/kainate channels and triggers selective neural injury.
AUTHOR: Yin H Z; Weiss J H
CORPORATE SOURCE: Department of Neurology, University of California, Irvine, 92717-4290, USA.
CONTRACT NUMBER: AG00495 (NIA)
NS30884 (NINDS)
SOURCE: NEUROREPORT, (1995 Dec 15) 6 (18) 2553-6.
Journal code: 9100935. ISSN: 0959-4965.
PUB. COUNTRY: ENGLAND: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199610
ENTRY DATE: Entered STN: 19961106
Last Updated on STN: 19970203
Entered Medline: 19961024
AB . . . Brief exposures of cortical cultures to kainate (100 mu M) plus Zn(2+) (300 mu M) cause fluorescence of the Zn(2+) **sensitive dye**, TS-Q, to appear in virtually all neurons, probably reflecting depolarization and secondary Zn(2+) permeation through **voltage-sensitive** Ca(2+) channels. However, if Na+ **ions** are removed from the media (to prevent depolarization), prominent TS-Q fluorescence is still observed in the small subset of neurons labeled by kainate stimulated Co(2+) uptake (Co(2+)(+) neurons), a histochemical technique that identifies neurons **expressing** Ca(2+) permeable AMPA/kainate receptor-gated **channels**. Kainate/Zn(2+) exposures in Na+ containing media with lower (50-100 mu M) Zn(2+) concentrations resulted 24 h later in selective loss. . .

L2 ANSWER 14 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN DUPLICATE 9
ACCESSION NUMBER: 1994:129924 BIOSIS
DOCUMENT NUMBER: PREV199497142924

TITLE: Cadmium Toxicity in Rat Pheochromocytoma Cells: Studies on the Mechanism of Uptake.
AUTHOR(S): Hinkle, Patricia M. (1); Osborne, Matthew E.
CORPORATE SOURCE: (1) Dep. Pharmacology, University Rochester School Medicine and Dentistry, Rochester, NY 14642 USA
SOURCE: Toxicology and Applied Pharmacology, (1994) Vol. 124, No. 1, pp. 91-98.
ISSN: 0041-008X.
DOCUMENT TYPE: Article
LANGUAGE: English
AB The uptake and toxicity of cadmium were compared in two rat pheochromocytoma cell lines: PC12 cells, which **express** **voltage-sensitive** calcium **channels**, and PC18 cells, which do not. PC12 but not PC18 cells responded to depolarization with an increase in 45Ca-2+ uptake and an increase in the concentration of cytoplasmic free calcium **ion**, (Ca-2+). These responses were blocked by the dihydropyridine calcium **channel** antagonist nimodipine and amplified by the agonist BAY K8644, drugs selective for L-type **channels**. Cadmium caused death of PC12 cells with an LC50 of 12 mu-M. Inclusion of high K+ with the agonist BAY. . . = 6 mu-M), whereas nimodipine protected against cadmium toxicity (LC50 = 30 mu-M). In contrast, drugs acting on L-type calcium **channels** did not affect Cd-2+ toxicity for PC18 cells (LC50 15 mu-M). Fura 2 was used to measure intracellular free Cd-2+. . . PC12 cells. Cd-2+ fluorescence appeared to be concentrated near the plasma membrane. The results confirm the potential involvement of calcium **channels** in cadmium transport and extend the use of intracellularly trapped fluorescent **dyes** to monitor intracellular free cadmium **ion** concentration.

L2 ANSWER 15 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 10
ACCESSION NUMBER: 1993:272255 BIOSIS
DOCUMENT NUMBER: PREV199396002480
TITLE: Neural induction suppresses early expression of the inward-rectifier potassium channel in the ascidian blastomere.
AUTHOR(S): Okamura, Yasushi (1); Takahashi, Kunitaro
CORPORATE SOURCE: (1) Dep. Neurobiol., Inst. Brain Res., Fac. Med., Univ. Tokyo, Hongo 7-3-1, Bunkyo-ku, Tokyo Japan
SOURCE: Journal of Physiology (Cambridge), (1993) Vol. 463, No. 0, pp. 245-268.
ISSN: 0022-3751.
DOCUMENT TYPE: Article
LANGUAGE: English
AB 1. Early **expression** of **ion channels** following neural induction was examined in isolated, cleavage-arrested blastomeres from the ascidian embryo using a two-electrode **voltage** clamp. Currents were recorded from the isolated, cleavage-arrested blastomere, a-4-2, after treatment with serine protease, subtilisin, which induces neural differentiation as consistently as cell contact. 2. The inward-rectifier K+ current increased at the late gastrula stage shortly after the **sensitive** period for neural inducation both in the induced (protease-treated) and uninduced cells. Ca-2+ **channels**, characteristic of epidermal-type differentiation, and delayed-rectifier K+ **channels** and differentiated-type Na+ **channels**, characteristic of neural-type differentiation appeared much later than the inward-rectifier K+ **channels**, at a time corresponding to the tail bud stage of the intact embryo. 3. When cells were treated with subtilisin. . . = 14) than in untreated cells (11.25 +- 3.10 nA, n = 26). The same changes in the inward-rectifier K+ **channel** were also observed in a-4-2 blastomeres which were induced by cell contact with an A-4-1 blastomere. However, when cells were. . . period for neural induction, the amplitude of the inward-rectifier K+ current was the same as in untreated cells. Thus the **expressed** level of the

inward-rectifier K+ **channel** was linked to the determination of neural or epidermal cell types. 4. There was no significant difference in the input. . . uninduced cells, indicating that the difference in the amplitude of the inward-rectifier K+ currents derived from a difference in the **channel** density rather than a difference in cell surface area. 5. The **expression** of the inward-rectifier K+ **channel** at the late gastrula stage was **sensitive** to alpha-amanitin, a highly specific transcription inhibitor. In both induced and uninduced cells, injection of alpha-amanitin at the 32-cell stage reduced the current density of the inward-rectifier K+ **channel** to about 2 nA/nF, corresponding to 13% of that recorded from uninjected cells. By contrast, the **expression** of the fast-inactivating-type Na+ current, which transiently increased along with the inward-rectifier K+ **channel**, was resistant to alpha-amanitin injection. 6. The dose of alpha-amanitin injected was controlled by monitoring co-injected fluorescent **dye**, fura-2. The dose of alpha-amanitin required for 50% suppression of the inward-rectifier K+ current was 3.0 ng/ml. This was close. . . was taken into account. 7. In the uninduced cells, injection of alpha-amanitin later than the 32-cell stage partially suppressed the **expression** of the inward-rectifier K+ **channel** and the fraction of suppression was related linearly to the time of injection. By contrast, in protease-treated cells (induced cells) the **expression** of the inward-rectifier K+ **channel** depended only on transcription before protease treatment. We concluded that inductive signals suppressed transcription of the inward-rectifier K+ **channel** which had already started before the 64-cell stage.

L2 ANSWER 16 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER: 1988:175601 BIOSIS
DOCUMENT NUMBER: BA85:87703
TITLE: FLOW CYTOMETRIC ANALYSIS OF MEMBRANE POTENTIAL IN EMBRYONIC RAT SPINAL CORD CELLS.
AUTHOR(S): MANDLER R N; SCHAFFNER A E; NOVOTNY E A; LANGE G D; BARKER J L
CORPORATE SOURCE: LAB. NEUROPHYSIOL., NATL. INST. NEUROL. COMMUN. DISORDERS STROKE, BUILD. 36, ROOM 2C02, NIH, BETHESDA, MD. 20892.
SOURCE: J NEUROSCI METHODS, (1988) 22 (3), 203-214.
CODEN: JNMEDT. ISSN: 0165-0270.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB. . . potential in suspensions of embryonic rat spinal cord cells was carried out in a fluorescence-activated cell sorter (FACS) using anionic **voltage-sensitive**, fluorescent **dyes** (oxonols). The FACS or flow cytometer is an analytical instrument that measures optical properties of large cell populations at a. . . is directly related to the degree of cell depolarization. Incubation of cells in elevated K+ concentrations or with the Na+ **channel** agonist batrachotoxin (BTX) changed the fluorescence intensity distribution pattern of the live-cell population; these changes were consistent with the depolarizing. . . in the dead-cell population. The BTX-induced shift was blocked by tetrodotoxin (TTX) and was reversed in Na+-free medium, indicating embryonic **expression** of functional Na+ **channels**. Fluorescence microscopy of sorted cells showed that live cells typically exhibited circumferential ring-like patterns, whose intensities were enhanced under depolarizing conditions. The results show that flow cytometry combined with oxonol **dyes** can be used to measure the relative membrane potential of large numbers of individual central nervous system cells. The analysis of the changes in the distributions of these membrane potentials can be used to reveal the development of functional **ion** conductance mechanisms.

L2 ANSWER 17 OF 18 EMBASE COPYRIGHT 2003 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 88006778 EMBASE

DOCUMENT NUMBER: 1988006778
TITLE: Flow cytometric analysis of membrane potential in embryonic rat spinal cord cells.
AUTHOR: Mandler R.N.; Schaffner A.E.; Novotny E.A.; Lange G.D.; Barker J.L.
CORPORATE SOURCE: Laboratory for Neurophysiology, National Institute of Neurological and Communicative Disorders and Stroke, National Institutes of Health, Bethesda, MD 20892, United States
SOURCE: Journal of Neuroscience Methods, (1987) 22/3 (203-213). ISSN: 0165-0270 CODEN: JNMEDT
COUNTRY: Netherlands
DOCUMENT TYPE: Journal
FILE SEGMENT: 002 Physiology
052 Toxicology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB . . . potential in suspension of embryonic rat spinal cord cells was carried out in a fluorescence-activated cell sorter (FACS) using anionic **voltage-sensitive**, fluorescent **dyes** (oxonols).
The FACS or flow cytometer is an analytical instrument that measures optical properties of large cell populations at a. . . is directly related to the degree of cell depolarization. Incubation of cells in elevated K+ concentrations or with the Na+ **channel** agonist batrachotoxin (BTX) changed the fluorescence intensity distribution pattern of the live-cell population; these changes were consistent with the depolarizing. . . in the dead-cell population. The BTX-induced shift was blocked by tetrodotoxin (TTX) and was reversed in Na+-free medium, indicating embryonic **expression** of functional Na+ **channels**. Fluorescence microscopy of sorted cells showed that live cells typically exhibited circumferential ring-like patterns, whose intensities were enhanced under depolarizing conditions. The results show that flow cytometry combined with oxonol **dyes** can be used to measure the relative membrane potential of large numbers of individual central nervous system cells. The analysis of the changes in the distributions of these membrane potentials can be used to reveal the development of functional **ion** conductance mechanisms.

L2 ANSWER 18 OF 18 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
DUPLICATE 11
ACCESSION NUMBER: 1981:183027 BIOSIS
DOCUMENT NUMBER: BA71:53019
TITLE: INTRA CELLULAR CALCIUM ACCUMULATION DURING DE POLARIZATION IN A MOLLUSCAN NEURON.
AUTHOR(S): GORMAN A L F; THOMAS M V
CORPORATE SOURCE: DEPARTMENT OF PHYSIOLOGY, BOSTON UNIVERSITY SCHOOL OF MEDICINE, BOSTON, MASSACHUSETTS 02118, USA.
SOURCE: J PHYSIOL (LOND), (1980) 308 (0), 259-286. CODEN: JPHYA7. ISSN: 0022-3751.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB The bursting pacemaker neuron R-15 of Aplysia [A. californica] was injected with the Ca2+ **sensitive dye** arsenazo III.
Changes in absorbance were measured with a differential spectrophotometer to monitor changes in free intracellular Ca2+ during membrane depolarization under **voltage** clamp conditions. **Dye** absorbance increased linearly for depolarizing pulse durations up to 100 ms and approximately linearly between 100-300 ms, but for longer durations the absorbance change decreased. The absorbance change vs. **voltage** relation increased steeply between -20 and 0 mV (e-fold/8.5 mV), peaked at +36 mV and declined nonlinearly to an estimated. . . null or suppression potential of about +139 mV. Tetrodotoxin (5 .times. 10-5 M) had no effect on the change in **dye** absorbance produced by brief or long duration stimuli whereas Ca2+ free [artificial seawater] abolished

all changes in **dye** absorbance. The absorbance change saturated with increasing external Ca2+ concentrations. The relation between **dye** absorbance and external Ca2+ concentration was hyperbolic and for a small range of external Ca2+ concentration and membrane potentials could be fitted by a Michaelis-Menten **expression** where the dissociation constant and the maximum absorbance change are **voltage** dependent. The absorbance change was reduced by external divalent **ions** which block the Ca2+ **channel** (e.g., Cd2+ and Ni2+). The suppression of **dye** absorbance was increased by membrane depolarization and suggests that there is a **voltage** dependent site within the Ca2+ **channel** which binds divalent **ions**. The decline of the absorbance-**voltage** relation from its peak to the suppression potential showed a greater nonlinearity when longer duration **voltage** clamp pulses were used. The nonlinearity can be explained if the accumulation of Ca2+ **ions** next to the inner surface of the membrane during depolarization reduces the driving force on Ca2+ **ions** decreasing Ca2+ **ion** influx. The suppression potential estimated from the absorbance-**voltage** relation increased 29 mV/10-fold change in the external Ca2+ concentration and can be used to estimate the Ca2+ equilibrium potential. The change in **dye** absorbance produced by brief depolarizing **voltage** clamp steps was inactivated at positive holding potentials (50% inactivation at about -14 mV). The slow decrease in **dye** absorbance during prolonged depolarization probably is caused by inactivation of the Ca2+ **channel**.

```
=> s (voltage (s) sensitive (s) dye) (p) ( recombina? (s) ion (s) channel)
L3            1 (VOLTAGE (S) SENSITIVE (S) DYE) (P) (RECOMBINA? (S) ION (S)
                CHANNEL)

=> d l3 total ibib kwic

L3   ANSWER 1 OF 1  BIOSIS  COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. on STN
ACCESSION NUMBER:   2001:417367  BIOSIS
DOCUMENT NUMBER:    PREV200100417367
TITLE:              Usefulness and limitation of DiBAC4(3), a voltage-sensitive
                    fluorescent dye, for the measurement of membrane potentials
                    regulated by recombinant large conductance Ca2+-activated
                    K+ channels in HEK293 cells.
AUTHOR(S):          Yamada, Aki; Gaja, Norikazu; Ohya, Susumu; Muraki,
                    Katsuhiko; Narita, Hiroshi; Ohwada, Tomohiko; Imaizumi,
                    Yuji (1)
CORPORATE SOURCE:   (1) Department of Molecular and Cellular Pharmacology,
                    Nagoya City University, Nagoya, 467-8603:
                    yimaizumi@phar.nagoya-cu.ac.jp Japan
SOURCE:             Japanese Journal of Pharmacology, (July, 2001) Vol. 86, No.
                    3, pp. 342-350. print.
                    ISSN: 0021-5198.
DOCUMENT TYPE:      Article
LANGUAGE:           English
SUMMARY LANGUAGE:   English
IT   Major Concepts
        Biochemistry and Molecular Biophysics; Pharmacology
IT   Chemicals & Biochemicals
        DiBAC4(3) [bis-(1,3-dibutylbarbituric acid)-trimethine oxonol]:
        limitation, usefulness, voltage-sensitive
        fluorescent dye; Evans blue: BK channel opener; NS-1619;
        recombinant large conductance calcium(II)-activated potassium
        ion channels
```